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PELLAGRA AND THE NEW DEAL*

By

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The work of Goldberger and his associates between the years 1914 and 1920 so firmly established the theory that pellagra was a disease due to dietary deficiency, that few serious efforts have since been made to disprove that proposition.

Probably the ablest contemporary opponents of this theory are Partlow and Tucker. Dr. W. D. Partlow, superintendent of the Alabama Insane Hospitals, believes pellagra to be an infection because of its regional distribution. He found it rare in Northern institutions in which the diet was practically identical to that of Southern institutions. He also observed that when first noted in this country it was more virulent than at the present time.(1)

Dr. B. R. Tucker, whose distinguished opinion cannot be lightly dismissed, believes that although vitamin deficiency is probably a contributing factor, the actual cause of the disease is a filterable virus which is probably allied to those of acute anterior poliomyelitis, influenza and herpes-zoster. He argues both from the regional incidence of the disease itself and from the distribution of the lesions in relation to peripheral innervation.(2)

In the papers cited, Tucker stresses the fact that pellagra reached its peak during the "fat" years previous to the depression and declined during the "lean" years which followed. With considerable justification he interprets this as indicating that the disease is not primarily a matter of dietary deficiency. The above phenomenon, although here differently interpreted, was what led to the writing of this paper, as will presently be shown. But whether we insist that vitamin deficiency is the primary cause of the disease or merely a contributing factor, there can be no serious doubt as to its immense importance.

We are all, of course, familiar with the clinical manifestations of the disease and likewise with the equally well known environmental factors usually associated with its development. We expect to encounter it among the submerged, or at best, the marginal sections of the population. It is chiefly a disease of the textile operative and the tenant farmer or share-cropper.

There are, it is true, many individuals not covered by the above classification who, nevertheless, develop this disease. Among these may be mentioned the fastidious person who refuses to eat certain foods, thereby bringing about a nutritional unbalance. There is the chronic alcoholic who does not assimilate his food properly, even though he may eat more than enough. There is the psychotic individual who is either indifferent to all food or who uses no judgment in his choice. There is the food-faddist who has allowed some self-styled "professor" to persuade him that man's salvation is by bran alone.

Nevertheless, statistically speaking, all the above are of rare occurrence with the exception of the psychotic patient who develops pellagra as a secondary condition. This group is fairly common in all mental hospitals, the psychiatrist being all too familiar with the manic-depressives, schizophrenics, involutionals and others

*Read before the Columbia Medical Society, June 8, 1936.

who have developed the disease upon the background of a primary mental disturbance.

But despite the occurrence of many such cases, it was the pellagrin per se who, in 1928, constituted 28.5 per cent of all admissions to the South Carolina State Hospital. It is this group to which the diagnosis, "Psychosis with Pellagra" is applied. This primary pellagrin is, as we have seen, drawn from an unfavorable environment, with negligible exceptions. He is the victim of undernourishment, particularly in regard to protein and vitamin deficiency.

In 1928 pellagra furnished one of the gravest problems confronting anyone attempting to treat mental disease. In that year the South Carolina State Hospital admitted 287 persons suffering from the disease and recorded 124 deaths due to its ravages. Of the latter 40 were white and 84 negroes, although admissions were almost equally distributed between the two races.

The next year witnessed little improvement, with eight more admissions and only three fewer deaths. Of the latter 50 were whites and 91 negroes. Nor were we at all encouraged when, in the autumn of 1929, the depression descended upon us. What prospects for statewide or sectional improvement might one expect in a disease whose very roots were supposed to spring from poverty.

Statistics for the following year (1930) are misleading, since they represent only nine months, a legislative act rendering necessary a change in the fiscal calendar. But it will be seen that the incidence of the disease was about the same, although the mortality was greatly decreased. During the nine months represented, 215 pellagrins were admitted and 71 died. Since the deaths were still recorded month by month it is possible, for purposes of comparison, to give the deaths for the calendar year of 1930, a total of only 87.

The figures for the next twelve month period, from September 30, 1930, to September 30, 1931, show 241 admissions and 80 deaths, of whom 13 are white and 67 colored.

By the following fiscal year, ending September 30, 1932, a sharp decline is noted in admissions as well as deaths. Of 129 admissions only 25 patients succumbed to the disease. Again, although racial distribution was about equal, as represented by the admissions, the deaths were in the ratio of four colored to one white.

With the next year the fiscal calendar was again changed by legislative act and the figures given represent only nine months, or from September 30, 1932, to June 30, 1933. During this time a total of 90 pellagrins received admission and 26 pellagrins died.

Fortunately for the rest of the period under consideration the statistics have been compiled for twelve month periods, each ending June 30. The next such period, from June 30, 1933, to June 30, 1934, shows 59 admissions with 23 deaths, white admissions and colored deaths again led.

For the year ending June 30, 1935, the figures are 54 admissions and 20 deaths. This is the last year for which complete figures are available.

Yr.	*Ad.*	*Died*	*Remarks*
1925	67	37	
1926	110	68	
1927	184	91	
1928	287	124	
1929	295	121	
1930	215	71	(January thru September)
1931	241	80	(Sept. 1930, thru Sept. 1931)
1932	129	25	(Sept. 1931, thru Sept. 1932)
1933	90	26	(Oct. 1932, thru June 1935)
1934	59	23	(July 1933, thru June 1934)
1935	54	20	(July 1934, thru June 1935)

The above figures are, I believe, rather significant, in that, while taken from a group, they constitute a fairly accurate cross-section of the general population of the State of South Carolina. For purposes of comparison, let me quote a few figures obtained from the State Board of Health, through the courtesy of Drs. James A. Hayne and Martin B. Woodward. The year by year incidence of the disease within this larger group, exhibits, as will become apparent, considerable similarity to that within the smaller group. In both instances it will be noted that 1928 and 1929 are peak years for deaths, though for morbidity 1930 is ahead of 1928 in the State Board's figures.

PELLAGRA STATISTICS FOR SOUTH CAROLINA
1925-1935 State Board

Year	*Reported*	*Died*	*Remarks*
1925		408	
1926	3053	529	
1927	4626	732	
1928	6409	937	
1929	7849	909	
1930	7311	613	(Nine months period)
1931	4827	637	
1932	3389	415	
1933	2489	353	
1934	1724	292	
1935	1991	310	

In relation to the above figures Dr. Woodward writes: "Unfortunately there was some change in the fiscal year in 1930 and again in 1933, so that there appears to be a certain overlapping in the periods covered. Nevertheless, I think it clearly evident that there has been a striking decrease in the number of cases during this eleven-year period."

After reviewing the figures above quoted it seemed desirable to obtain the statistics for the previous decade for purposes of comparison. The State Board again cooperated by furnishing its mortality figures, which are given in the following table. No morbidity figures were kept at that time. It will be noted, however, that most of these are for ten month periods, only two being twelve month periods and one eleven month. Nevertheless, it is very apparent that in 1915 there was an enormous number of deaths due to pellagra, the rate declining about 60 per cent the next year. Except for a few minor rises, it gradually goes down from 1916 to 1925, when there is another sharp rise, but nothing to compare with the figure for 1915, the first year statistics were kept by the State Board.

Again these figures bear a close resemblance to those of the State Hospital, whose complete statistics are presented below. Following the independent discovery of the disease at the South Carolina State Hospital in 1908, a statewide conference was held, followed by a national conference in 1909. Monthly figures are available from 1908 for the State Hospital, showing that by 1910 the disease had reached epidemic proportions. 1914 and 1915 were the peak years with the remarkable drop, already noted, in 1916. With regard to this drop Dr. Woodward writes:

"Between 1912 and 1915 an extensive Educational Campaign was used with the result of better diagnosis being made. This, together with the fact that by 1915 the so-called epidemic was probably on the wane, explains why there was such a drop of the deaths from 1915 to 1916."

ADMISSIONS AND DEATHS DURING FISCAL YEAR
STATE HOSPITAL

Year.	*White Adm.*	*White Died.*	*Col. Adm.*	*Col. Died.*	*Total Adm.*	*Total Died.*
1908		4		29		33
1909		16		52		68
1910		26		80		106
1911		50		110		160
1912		95		133		228
1913		66		99		165
1914		136		220		356
1915		96		231		327
1916	108	59	100	82	208	141
1917	101	45	73	67	174	112
1918	87	59	56	66	143	125
1919	34	19	28	24	62	43
1920	21	15	62	14	83	29
1921	39	15	54	39	93	54
1922	45	17	54	38	99	55
1923	39	23	61	45	100	68
1924	34	16	48	54	82	70
1925	48	21	19	16	67	39
1926	67	29	43	39	110	68
1927	107	34	77	57	184	91
1928	145	40	142	84	287	124
1929	149	30	146	91	295	121
1930	97	17	118	54	215	71
1931	121	13	120	67	241	80
1932	63	5	66	20	129	25
1933	55	10	35	16	90	26
1934	37	7	22	16	59	23
1935	33	9	21	11	54	20

Remarks

1908 No figures available for admissions
1928 28.5 per cent of total causes of death
1929 28.47 per cent of total causes of death
1930 First nine months
1933 First six months

PELLAGRA STATISTICS FOR STATE OF SOUTH CAROLINA BOARD OF HEALTH

Year.	*Reported.*	*Died.*	
1915		1379	January-November
1916		553	January-October
1917		544	January-October
1918		558	January-October
1919		359	January-December
1920		219	January-October
1921		260	January-October
1922		319	January-October
1923		267	January-October
1924		275	January-October
1925		408	January-December
1926	3053	529	January-December
1927	4626	732	January-December
1928	6409	937	January-December
1929	7844	909	January-December
1930	7311	613	Jan.-Sept. 9 mos. period
1931	4827	637	January-December
1932	3389	415	Oct. 1, 1931-Sept. 30, 1932
1933	2489	353	July 1, 1932-June 30, 1933
1934	1724	292	July 1, 1933-June 30, 1934
1935	1991	310	July 1, 1934-June 30, 1935

Now the feature of interest in these figures lies chiefly, as I see it, in the fact that this disease, in which we would have expected a marked increase, has shown a steady decline during the very worst depression years.

This phenomenon is by no means confined to our own State. Reece called our attention a year ago to the fact that 1928 was the peak year for pellagra in Alabama, Kentucky, Mississippi and South Carolina, whereas Virginia, Louisiana, Georgia and Florida reached their worst year in 1929. He attributes this fact in part to the floods of 1927 which affected Arkansas, Tennessee, Mississippi and Louisiana.(3) This, however, would not account for the increase in South Carolina. The same observer noted the universal decline beginning in 1930.

For the entire registration area the death rate per 100,000 was 5.0 in 1927, 6.1 in 1928, 5.8 in 1929, 5.3 in 1930, 4.3 in 1931 and 3.1 in 1924.(4)

In attempting to account for this decline during the depression years many factors have to be considered, among which may be mentioned general educational enlightenment, public health propaganda and the widespread adoption of brewers' yeast both as a preventive and a therapeutic measure.

Another element of undoubted importance is the greater dependence of man upon the soil in times of industrial insecurity. Many families who formerly depended upon textile work alone found time in the slack years to grow a garden or care for a cow. Though their clothes might suffer, their stomachs benefited.

But to this observer, at least, it does not seem apparent that the factors discussed above are sufficient to account for the sharp drop in 1932. It is worthy of note that this is the year during which that social and political upheaval known as the New Deal came into being.* A precipitous decline in the pellagra figures was immediate. Is it not, therefore, reasonable to conclude that, in the free distribution of foodstuff, the creation of new jobs under the various government relief agencies, the CCC camps and the direct relief afforded by the administration, that we have the answer? I think that the figures show too close a relation to the New Deal for us to disregard them. It is true that 1928 and 1929, the worst pellagra years, were the period when two cars were supposed to repose in every garage and a chicken in every pot, but they were also the years when money crops were raised to the exclusion of food crops and when earnings went not into intelligently selected food but into vacuum cleaners and radio sets.

That the decline, already begun in 1930, was tremendously accelerated by the New Deal, seems indicated by the comparative death rates from 1927 through 1932 in the neighboring commonwealths of North Carolina and Mississippi, both typical pellagra states.

Year.	*North Carolina.*	*Mississippi.*
1927	22.8	35.4
1928	28.0	38.0
1929	30.4	36.6
1930	31.2	28.5
1931	21.5	26.6
1932	15.0	15.0

*While it is true that the various relief agencies did not go into effect until 1933, the New Deal really began with the 1932 elections.

This paper is not intended in any sense as a commentary upon the New Deal. I have no quarrel whatever with those who may think certain of its activities unconstitutional or unwise. My purpose is merely to call attention to a scientific fact and to place upon it my honest interpretation. That interpretation is that pellagra, already on the wane after 1929, received a staggering blow as a result of the government's effort to provide the forgotten man with the necessities of life.

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SCREW WORM INFESTATION

By
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Screw worm infestation in South Carolina is principally a problem for the veterinarian and the economist. But here, as in other new territory which it has invaded, there are enough cases of human infestation to demand the attention of the medical profession.

This pest has been known in the Central American countries for many years and from there it spread up into the great southwest of our country. In the last few years it has traveled along the gulf states and up the Atlantic Coastal region. In 1934 the fartherest north a case of animal infestation reported in this section was near Ridgeway, S. C. In 1935 cases were being reported around Fayetteville, N. C. The spread will continue northward until climatic conditions become unfavorable for its cycle of development.

Read before the South Carolina Medical Association, Greenville, S. C., April 23, 1936.

There is a notion that the big influx of cattle into South Carolina in 1934 from the scorched plains of the west is the cause of its rapid appearance over the state. But this is not the case. A veterinarian, who inspected thousands of these cattle, injured during the hot weather by shipping accident, said he did not see a single case of screw worm infestation. So this is one misfortune that can't be laid at the door of the "New Deal."

The eggs of screw worm are laid by a certain fly (Cochliomyia americana) which belongs to the blow fly family. It is bluish green with three stripes along the back between the wings and with a yellowish red face. This fly deposits great numbers of yellowish eggs in irregular masses on the edge of cuts, abrasions, blood spots or wounds of any kind. With favorable temperature these eggs will hatch in the remarkably short time of twelve to fourteen hours. The young worms promptly begin to penetrate the tissues causing pain and a serosanguineous discharge of a most disagreeable cadaverous odor. In young animals these eggs are sometimes deposited around the navel or sometimes around the teeth as well as in all sorts of artificial wounds. In the human they are sometimes deposited in the nose where there is a nasal discharge or some small ulcer. These worms grow rapidly and reach about the length of 1-2 to 3-4 of an inch. After four or five days the adult worms drop out of the wound and bury in the ground and after a few days there emerge as the fly which soon mates and more eggs are deposited.

There is very little liklihood of an infestation in a normal vigorous person but in childhood or old age or the debilitated where deep sleep or stupor dulls the sensibilities. The person with cancer, old ulcers or osteomyelitis on the exposed areas of the body, who are unprotected during sleep furnish an inviting field. The child with impetigo is also susceptible under the same condition.

As the young worms begin their penetration and destruction of live tissue there is produced

a burning, pinching pain and characteristic wine colored sero-sanguineous discharge and characteristic odor, referred to as cadaverous.

The animal or person quickly takes on a septic or "sick" appearance and usually results in death if not given proper attention. In the human there is a considerable rise in temperature and increase of leucocytes and chilly sensations as would be expected in such toxic wounds.

One thing peculiar about this worm is that it does not bury itself completely in the tissue and therefore it is very seldom that enlargement or cutting is necessary in treatment. They crater out the wound by destruction of tissue always eating in with the sharp or pointed end, which has two strong rather powerful hooks or jaws which aid in tearing the tissue. The blunt or rear end remain in contact with air, as the breathing apparatus is situated there and appears as two brownish plates. The worms lie usually side by side, arranged somewhat analogous to cavities in honey comb. There seems to be a slight rotary motion as the worms busy them-selves with their destructive feeding. Their motility is due to the circular spine around the body which are similiar to the thread on screws and from which it gets its characteristic name.

The symptoms are so characteristic that when this condition is placed on your ever-increasing list of probable diagnoses, a correct diagnosis is very easy. In easily accessible wounds the worms are readily seen. In the nasal cavity when they develop rather high up the view may be obscured by partially dried secretion. After a cleansing solution the nasal speculum brings them well in view.

The prophylaxis depends on the proper protection of all wounds from the possibility of contact from flies and proper screening of young, old and debilitated especially during sleep. Also measures should be taken to control the development of flies by destruction of worms removed from wounds and the burning of all animals who die as the result of infestation.

The treatment is also rather simple. It consist of mopping away the secretions with gauze or cotton, cleansing with peroxide of hydrogen and irrigating with Boric acid solution or 1 to 2000 potassium permanganate solution. This is done to reduce the disagreeable odor and to remove the secretions which attracts the flies and encourage another deposit of eggs. Most of all these measures are carried out so that the real destructive agent can be applied. Two very effective agents are had in chloroform and benzol, the latter being in more universal use. The wound is lightly packed with gauze saturated with benzol so that the fumes are inhaled by the insects. The inhaled fumes are equally as effective as actual contact of the drug with the insects.

Briefly the patient that prompted this presentation was a farmer 72 years old who lived in an unscreened house and who during the summer took naps at the noon hour either on the porch of his house or on the ground in the shade of the trees. He said he had had "catarrh" for quite a while, otherwise no disability. Was somewhat debilitated on account of age and a rather limited food supply. Five days before seen felt chilly and a little weak. He noticed that his accustomed exercise tired him, thought he had some fever. Noticed a little bloody discharge and which he thought was nose bleed and it was for the last condition that he sought relief. The first impression was a malignancy of the nasal structures. After some Ephedrin solution was dropped into the nose to constrict the tissues and stop the oozing, the worms were brought into view. The nose seemed packed with worms lying side by side. A few worms were dislodged. The following day benzol was instilled into the nose and very readily 20 worms were blown out. For several days following, the nares were lightly packed with gauze saturated with benzol and over 200 worms were gotten out of right side of nose. There was no infestation on left side. During this time efforts were made to have him hospitalized which was finally done after several days delay but no worms found after admission to hospital. There was a great deal of destruction of mucuous membrane over septum, turbinates, etc. With irrigations of saturated boric acid solution and instillation of 100 per cent neo silvol solution the wound granulated as rapidly as could be expected. He was discharged after three weeks stay in hospital in a fairly satisfactory condition.

DISCUSSION

Dr. E. A. Hines, Secretary, Seneca:

Dr. Wallace was kind enough to give me a copy of his paper a few moments ago to look over. It is obvious to you that I do not know anything about the real subject presented. The Program Committee, however, looked upon this paper as being one of the most important that we have at this session, and I believe that you will think so, too. Dr. Wallace does not appear upon our program often, but he is a real investigator, and every time he appears he makes a real contribution. He was one of the first to make a study of pellagra in South Carolina, one of the first states in the Union where extensive research on this disease was made. Dr. Wallace presented an epoch-making contribution a few years ago on tularemia, being one of the first in this country to do so. Now he appears with another paper of which I know you will be proud. Dr. Wallace is also a true physician, for he has his mind always on preventive measures.

He has been thinking of the vast number of people who may be affected by this infestation—first, the children; second, the aged; third, the feeble.

Dr. W. P. Timmerman, Batesburg:

I should like to mention the fact that Dr. Wise, of Saluda, several months ago presented a case similar to that of Dr. Wallace. Dr. Wise, through his timidity, did not write it but presented it orally to the local medical society.

I wish to congratulate Dr. Wallace.

Dr. James A. Hayne, State Health Officer, Columbia:

I simply wish to call to the attention of the medical men in that State that we have something in the State that we have had no record of having before up to last year—that is, fatal infestation of a human being by screw-worm larvae. In looking over my records I found eight cases of fatal screw-worm infestation. Now, remember that last year was the first year that screw worm made its appearance among animals to any great extent. We have, as you know, the black blow-fly, we have the green blow-fly, and we have this screw-worm blow-fly. Very characteristic of this fly is that it has a red head. The other flies that you know do not have red heads, neither the common-house-fly nor the green blow-fly nor the black blow-fly. This fly has three white stripes on its back. It will lay its eggs whenever it can find fresh blood, and it will come from a long distance to find fresh blood. It has been likened to the buzzard, which may not have been seen at all until carrion appears, when it is seen coming down out of the empyrean blue. The danger to human beings is the rapidity with which this fly lays its eggs. This screw fly lays its eggs rapidly, one after another. The egg hatches within four hours, under favorable circumstances, after being laid, and develops a maggot or screw worm, which very rapidly increases in size and in less than five days becomes a pupa and drops out of the wound. The fly can lay from 255 to 300 eggs.

The worms may not be seen in the cavity unless you look for them. In the case reported by Dr. Wallace he did not suspect screw worm, and had no idea it was there. He looked within the nostril of the patient with a flashlight, saw the movement there, and thought it was the pulsation of the artery. I mention that because we might not think to look for screw-worm infestation. Of course, the treatment is easy; 90-per-cent commercial benzol is applied to the wound, then apply a pledget of cotton or gauze.

The prevention of screw-worm infestation is simple, of course. Paint fresh wounds with pine oil. The odor of the pine oil repels the fly.

The cases of this infestation are easy to find if we look for it, but we shall not find it unless we look for it.

I live on a farm and have eleven hogs, three calves and one goat on the place infested with screw worm The calves were infested at the umbilicus; the hogs were infested when castrated. Now, in the West they do not have hogs castrated and do not allow cows to have calves from May until heavy frost, because they know the animals will be infested in that way. It is a fatal disease for animals because of the weakness from infestation and the toxemia produced from entrance of the worms in great numbers.

Dr. Wallace, closing the discussion:

I have nothing further to say except that I appreciate the discussion by these gentlemen. I simply wish to say that if the little effort I put forward in the preparation of this paper aids in the prevention of one single case of this infestation or in the cure of one case of this horrible condition I shall feel amply repaid.

TYPHUS FEVER

By

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Typhus fever is a member of a family of maladies which are grouped together under the name of Rickettsia diseases. The name was given in honor of Ricketts, an American, who died of typhus while studying the disease in Mexico City. The particular variety which is responsible for typhus is called "Rickettsiae Prowaceki" adding the name of Prowacek, an Austrian who died in the same manner. These minute bacillus like things acquired their first parasitism on insects. Similar organisms in-

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Read before the Ridge Medical Society, Batesburg, S. C., April 20, 1936.

capable of producing disease in man have been found in a variety of insects. Parasites of this order have been found in sheep lice, in dust lice, bedbugs, mosquitoes, mites and ticks. The Rickettsiae needed a name for themselves because they could not be logically grouped with either the bacteria or with the protozoa. Now it seems that the two hosts involved in giving typhus to man are the louse and the rat flea on which are parasitic the Rickettsia. The infected louse dies within two weeks; but the infected rat flea gets rid of the parasite in a month or two and recovers. If one or more of these infected fleas or lice feed on man, they transmit typhus fever. The louse is the more dangerous and causes Epidemic Typhus because it stays with it's host so persistently until it (the louse) dies. While a flea does hop away at times, Epidemic Typhus fever, as seen along the Southeastern Coast is known as Brills disease. Brill first described it in 1898 among the Jews of New York. He thought it was a new disease but it was later proven to be true typhus brought to this country by immigrants from Southeastern Europe who had previously had the disease, some even in childhood and in whom the virus had lain dormant for years and then had another attack or recrudescence, which would under conditions of louse or flea infestation spread the disease.

The fifteenth century is as far back as typhus fever can be authentically traced, altho it may have existed for thousands of years and may have been among the many epidemics of the remote past which killed millions and were known as plagues. And just to think that most of the work in discovering the facts about typhus has been done since 1928, and some of the most recent investigations are not yet in print.

Now the prevalence of this disease in our own section of the country may be noted by the comparison of the number of cases reported in other sections. There were 528 cases in New York City and Boston from 1900 to 1930. In South Carolina 1927-1935—9 years—259 cases. In 1933 there were reported in Georgia 637 cases.

During the world war I witnessed the delousing processes on soldiers and clothing but was surprised to recently read a report of the typhus situation on the far Eastern front. It said typhus began in the Serbian Army. There were less than 400 doctors in the Country, almost all of whom contracted the disease and 126 died. The new cases per day ran into many thousands—2500 were admitted daily to the military hospitals alone. In less than 6 months over 150,000 people died of typhus. This was in 1914. The Serbs had 60,000 Austrian prisoners and one half died from typhus. In Russia the prevalence and ravages of typhus during the war reads like medieval history. During the war and until 1921 there were no less than 30 million cases of typhus with three million deaths in Soviet Russian territory. The remarkable thing is the total absence of typhus from the Western front. Extraordinary sanitary measures, bathing and delousing in the Allied as well as the German armies must be given the credit. The Germans would not transfer their troops from the Eastern front to the Western front for fear of bringing typhus which they knew would cause them to lose the war.

My interest in this disease was aroused by the fact that I had under my care this year 4 cases at one time, the 4th case going down with his invasion about one month after the first case started. Two cases were in the same household, husband and wife. The wife going to bed about two and a half weeks after the husband's invasion. The third case was in the City limits but widely separated and no contact. The 4th case was in the Southern section of the town and no known contact with the other cases. These cases were diagnosed finally as Brill's disease or Endemic Typhus Fever.

The first case I diagnosed as Influenza and waited for the usual symptoms to develop to confirm the tentative diagnosis. These symptoms did not develop. The patient was apparently very ill and I watched him closely. In my routine examination on the fifth day I observed a mascular rash over his chest and arms and abdomen which looked very much like rose spots and on pressure they would fade and return. The blood tests were negative to typhoid and paratyphoid. I remembered two years previously having seen at the same time two cases which had a similar rash and similar course of clinical symptoms which I

diagnosed as Typhus Fever. Both cases were connected with Feed Stuff Stores and I was told at the time by a Public Health Expert that we could always connect these few Endemic cases with such a contact. My impression at that time was that the disease was produced by the bite of the infected fleas from the Norwegian wharf rat and that this flea was transferred in the feed stuff in interstate commerce. Now I am told that the final step forward in the etiology of this disease as made by the United States Public Health Service, is that there exists in nature a permanent rodent reservoir for Endemic Typhus Fever in the rat and that the disease is transmitted to man thru the feces of infected rat fleas. As the flea bites he at the same time defecates. The person in rubbing the flea bite inoculates himself with the infected feces. This to my mind is the only way to explain my four cases.

From all historical accounts old world Epidemic Typhus Fever was a terrible scourge. In old English History during Tudor times there were great epidemics of typhus fever and it was then considered to be a disease of filth, war and misery. One historian (McLaurin) says probably if we could get at the real truth, throughout recorded history that Typhus had slain more people than any other Epidemic disease. It was then spoken of as the "Sweating sickness." It seems to have begun with the war of the roses and when Henry VII returned to London in triumph after the battle of Bosworth with his Typhus infected army and it's millions of fleas and rats the disease spread among the over crowded houses of the capital and high and low perished alike. Nobody seemed immune. In one week it took two Lord Mayors and six Aldermen and the Ancient University of Oxford closed it's doors and professors and students alike fled in common terror.

Sir William Osler in his 1910 edition of Practice of Medicine says Typhus Fever is an acute infectious disease of unknown origin. He quotes Hirsch as saying, "The History of Typhus is written in those dark pages of the world's history which tells of the grievous visitations of mankind by war, famine and misery of every kind." In England in 1875 there were one thousand four hundred and ninety nine deaths, in 1895 only 58 deaths. In 1897 there were only three cases of Typhus Fever in the London Fever Hospitals. Still Osler in his time considered it a highly contagious disease, stating that among 1230 physicians attached to institutions in Ireland 550 succumbed to this disease. Medical science has achieved a triumph in the disappearance of the epidemic form in most countries and in the intensity of the symptoms and the reduced mortality. All this improvement evidently due to better sanitation and a correct knowledge of the source of infection.

During the time that I was treating these four cases, I was very fortunate to receive a reprint from the Georgia Medical Association Journal by Dr. Mark S. Dougherty, Jr., of Atlanta, Ga. on Endemic Typhus Fever in Georgia. He says a review made of the charts of all patients dismissed from the hospitals in Atlanta with the diagnosis of Typhus fever from 1925 to January 1, 1934, shows the number to be 69. Of this number nine cases were doubtful and not included in his study. In 1933 six hundred and thirty seven cases were reported in Georgia. The disease seems to be more prevalent along the Coastal Country and radiate from port towns. For a five year period Atlanta reported 96 cases and Savannah, Ga., 339 cases. My four cases followed so closely the usual clinical course of the disease that I will not report the cases separately but will mention only the general course and any special developments. None of my cases were in the hospitals and I had to depend on home nursing and no regular records were kept, by a professional nurse. Only one case complained of prodromal symptoms and he said he had suffered headache and backache and general malaise for about a week. In all the cases the onset was sudden with a rapidly rising fever, chills, severe headache and some nausea. The headache was the most complained of symptom. In one of my cases it was so severe that I had to eliminate the thought of meningitis. Medicines don't seem to give much relief for this headache. All cases showed a complete anorexia. There was great prostration, insomnia and nervousness. One case had profuse sweats and I left off all antipyretics but I found these did not cause them but they were due to the disease. When the epidemic type was rampant

in England they called it the "English Sweat." The temperature reaches it's height about the 5th day. All cases showed a temperature of 104 degrees at some time during illness. The temperature would vary one to three degrees during 24 hours. Would run around 101 1-2 in the morning and then 103 1-2 to 104 in the afternoon. The rash is the most distinctive thing about the disease. It comes out from 4th to the 6th day and lasts several days. It shows up first on the lower chest and abdomen and then on the back. If the skin is hot it shows better. Some of it will fade away if the room is cold and the cover removed for examination. It spreads to the shoulder and inner surface of the arms and thighs. The rash consists of rose to dull red macules. One of my cases, the female, had a peculiar form of the rash which I could only find described in Osler's work. On the fifth day I could see it plainly under the skin just like looking through a film of oil and I promised her the rash would be out next day. It never did show up any plainer. Osler described this rash when he said it had the appearance of a "fine, irregular, dusky red mottling as if below the surface of the skin, some little distance, and seen through a semi-opaque medium." My first case had been free of fever three days when he was taken with severe pains in his left leg and suffered all night. He reported to me next morning that he had suffered with "Cholly Hoss." On examination I found that he had suffered a thrombus and was kept in bed several weeks longer by this complication. One patient, third case, was normal three days and then suffered a relapse that lasted about one week. The fever lasted in all four cases 14 days and all came down by lysis.

The Weil-Felix test was negative in all cases on first specimens of blood sent to the laboratory. Later in the disease I got a positive report in two of the cases in dilutions -1-50, 1-100-1-200-1-400. In the other two cases I sent the second specimen and it came back negative. This agglutination test is not of much aid in diagnosis unless made daily, as it may be negative one day and completely positive the next day. The cases that were negative to the test had the same clinical course and symptoms and the same disease that the two positive cases had. When the fever left the patients they felt better and convalescence was rapid in the uncomplicated cases.

Endemic Typhus occurs sporadically. The mortality is 1 to 4 per cent. It is not connected with filth or over-crowding and no respecter of persons or social status. Incubation period 7 to 14 days. It is claimed now that there is no evidence that it spreads from man to man. There are several contagious and infectious diseases with rashes from which it is necessary to differentiate. As all the books give a complete list of these I will not burden you. The main things for us, as Doctors, is to know that we have this disease with us yearly and be on the alert to recognize it, and as men with two eyes and a brain behind them to see if we can ferret out the information that may help to prevent the disease entirely.

THE SIMPLIFIED KETOGENIC DIET IN THE TREATMENT OF BACILLI INFECTION OF THE URINARY TRACT IN GENERAL PRACTICE

By
J. H. CUTCHIN, M.D.
Easley, S. C.

This subject is nothing new, but the object of this paper is to present a method by which a treatment of bacilli infections of the urinary tract can be carried out in general practice, and to show the simplicity of the treatment, so that it may be carried out in one's office.

It is offered as a relief for a great army of patients who suffer from these infections and who have not got relief from the usual methods of treatment.

We may consider that around eighty percent of all urinary infections are due to the colon bacilli(1) (Crane). This treatment is not intended to replace the surgical treatment of perinephric abscesses, obstructions of the ureter, or calculi, although some men have claimed to get relief by the use of this diet in certain cases pyelonephritis and infections caused by the presence of calculi.(2)

This diet is primarily intended for adults,

Read by title before the South Carolina Medical Association, Greenville, S. C., April 22, 1936.

but can be used in cases of children with pyelitis or cystitis.

The diet may be prescribed without detailed dietetic knowledge by the physician and can be prepared in the average home without special dietetic supervision.

The menus are so arranged that regardless of the items collected from the different groups, the daily intake of food will consist of carbohydrates, 15 to 20 grams; proteins, 35 to 50 grams; and fats, 300 to 325 grams. The vitamin deficiency need not be considered here as the patient is only kept on the diet for a short time. The menu also instructs the patient in detail how to choose his diet, and any patient of average intelligence can follow it.

The detailed chemical changes which this diet covers will not be discussed, owing to the limited time, but two conditions must be present in order that we get the best results with its use. First, sufficient concentration of ketone bodies, second, coincidential with this, an acidity of the urine of a ph. of below 5.3, as the successful use of the ketogenic diet is dependent on the bacteriostatic or bactericidal action of beta-oxybutyric acid at a certain degree of acidity.

Let us now look at the diet that will produce this ketosis. I will not read the entire diet, but will name a diet for one day, in order to show the type used. A copy must be given to each patient, so that he may work it out. The diet is as follows:

The Simplified Ketogenic Diet

GROUP A—EGG DISHES

1 Egg nog
Egg—1
Cream (40 per cent fat)—6 tablespoons
Water—5 tablespoons
Nutmeg—to taste

2 Scrambled eggs
Eggs—2
Cream (40 per cent fat)—7 tablespoons
Butter—3 teaspoons

3 Egg omelet
Eggs—2
Cream (40 per cent fat)—7 tablespoons
Butter—3 teaspoons

4 Egg custard
Eggs—2
Egg yolk—1
Cream (40 per cent fat)—7 tablespoons
Vanilla—few drops

GROUP B—SALADS

1 Lettuce salad
Lettuce—1-8 head
Mayonnaise—4 tablespoons

2 Lettuce and tomato
Lettuce—1-8 head
Tomato—1 small
Hardboiled egg—1 yolk
Mayonnaise—5 tablespoons

3 Combination
Lettuce—few leaves
Celery—2 hearts
American cheese (grated)—2 tablespoons
Mayonnaise—4 tablespoons

4 Asparagus
Asparagus—6 stalks
Lettuce—few leaves
Mayonnaise—4 tablespoons

5 Egg salad
Lettuce—few leaves
Egg (deviled)—1
Mayonnaise—4 tablespoons

GROUP C—CREAM DESSERTS

1 Bavarian Cream
Gelatin—1 teaspoon
Cream (40 per cent fat)—7 tablespoons
Whip the cream. Soak the gelatin in 1 teaspoon of cold water. Dissolve in 2 teaspoons of hot water. Add 2 drops of any flavoring. When cooled, add to cream. Place in mold and chill.

2 Gelatin
Make plain gelatin as in No. 1. Use 7 tablespoons of unsweetened whipped cream over it.

3 7 tablespoons of whipped cream, with or without flavoring.

GROUP D—BEVERAGES

Tea, coffee, or water, with 4 tablespoons of cream. If desired, this amount of cream may be used with two cups of beverage. Use no sugar or milk.

DAILY MENU

Breakfast—

1. Choice from Group A.
2. One choice from Group D.
3. 8 slices of thin, crisp bacon, or 4 tablespoons of cream (40 per cent fat).

Dinner and Supper—

1. One choice from Group B.
2. One choice from Group A or C.
3. One choice from Group D.

DIRECTIONS

1. Satisfactory results cannot be obtained unless this diet is followed as outlined. Even the smallest deviations may ruin the chance for success.
2. No food or beverage other than that listed may be taken.
3. Water may be taken in moderate quantities, as desired.
4. The chewing of gum or tobacco is not permitted.
5. No laxative other than that prescribed by the physician is to be taken.
6. Do not take any medicine other than that prescribed by the physician.

In the majority of cases a ketosis will develop in three to five days and the patient who develops a ketosis quickly will be more benefited than the one who develops it slowly. In those cases that do not develop a ketosis quickly, a glass of 40 per cent cream may be taken between meals; then if the patient does not develop the ketosis, you may suspect that he is not carrying out the diet.

Some patients will have a certain amount of nausea and a feeling of weakness while on the diet, but this is to be expected in some cases. Normal work and exercise may be carried out during the treatment. Water is not forced, but a moderate amount may be taken.

Several tests have to be run each day. First, daily tests of the urine for evidence of the ketosis, which is based on the test of diacetic acid in the urine. The test is run as follows: to an equal part of 10 per cent aqueous solution of ferric chloride add the same amount of the patient's urine. If a ketosis is present, a port-wine color will result. The second test, which should be run, is to determine the ph value of the urine, which should be below 5.5, but I have seen cases in which the ph was higher and still an improvement was noted. In order to determine the ph value of urine, a method is used which can be done in the office. To twenty drops of the patient's urine, add one drop of 0.04 per cent solution of chlorophenol red. If the color of the urine remains the same, it is safe to assume that the acidity is around ph 5.2. The pinker it turns, of course, the higher the acidity.

Microscopic study of the urine should be made every other day to see whether pus or bacilli are present.

The use of drugs in this treatment is varied. I will mention only the ones I have used in my cases. These were used only after an appearance of the ketosis. In those cases in which the acidity of the urine was above ph 5.2 ammonium chloride in the form of enteric coated tablets was given in the amount of 80 to 90 grains daily until the ph was lowered. The use of methenamine may help in some particular cases. Colon bacilli made by Krueger method were used in all of the cases. This is given hypodermically, as any bacterial antigen. No other drugs were allowed. Daily soap suds enemata were taken by each patient. Bacillus Acidophilus was given each day to change the intestinal flora.

Patients should not be kept on this diet for more than ten or twelve days, whether the results have been favorable or not. Several short courses of the dietary treatment are preferable to one long course. If microscopic examinations do not reveal the organism for three consecutive days, the diet should be discontinued.

There will be cases in which we do not get satisfactory results. In those cases we should search for some foci of infection, such as an involved prostate gland, urethral cysts, or infected urethral glands. When the prostate gland is found to be infected, gentle massage of the gland is given every other day.

In some cases where the cocci type of infection is associated with the bacillary type, neo-arsphenamine gives results.

There are two other types that do not respond to treatment and no explanation can be given for this. These are the group which will not develop a ketosis, and the one in which the ph

of the urine remains high in spite of the administration of large doses of some acidifying agent.

SUMMARY

1. From the literature and in my experience, we have a valuable addition in the treatment of cases of cystitis and pyelitis caused by the colon bacilli.

2. Owing to the simplicity of the treatment, every physician in general practice should be able to carry the treatment out in his office.

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SOUTH CAROLINIANA

J. I. WARING, M.D., CHARLESTON, S. C.

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SEPTEMBER, 1936

AMERICAN BOARD OF INTERNAL MEDICINE

One of the most far reaching developments in American Medicine, in all probability, is that of the new Board designed to certify specialists in Internal Medicine which begins to function actively with the first written examination in December, 1936. Successful candidates at the written test will be eligible for the practical and clinical tests in April, 1937. The fee for the examination is forty dollars. Application blanks may be secured by writing to Dr. Walter L. Bierring, 406 Sixth Avenue, Des Moines, Iowa.

It is pretty well conceded that a broad foundation in internal medicine is highly desirable for any medical student or physican, regardless of the specialty he may intend to pursue. The well known divisions of medicine and surgery have for a long time been recognized, and certain organizations like the American College of Physicians and the American College of Surgeons have done much to bring about higher standards of qualification. The general plan of this Board is not very different from that of some eight or ten other Boards of certification for specialists already established. In looking over the requirements one is impressed with the high standard to be demanded of the candidate.

This Board is an outgrowth of the activities of the American College of Physicians and the American Medical Association along this line. The examiners include men of the highest eminence in both the United States and Canada. The Certification Board for Surgeons is to be organized shortly also.

FOUNDER'S DAY AT THE MEDICAL COLLEGE, NOVEMBER 5

An important event every year in our State now is that of the celebration of Founder's Day at the Medical College of the State of South Carolina. The exercises usually include an address by an outstanding clinician, and this year it is a pleasure to announce that this man will be Dr. Reginal Fitz of Boston. The mere mention of his name brings to our minds the enviable traditions of American Medicine. More details will be given later, but the event should be looked forward to and placed on the calendar of every physician in South Carolina as one of importance to our State.

THE POST GRADUATE COURSE AT ANDERSON

As was anticipated, the Clinical Assembly held at Anderson was a marked success. About one hundred physicians were in constant attendance. The officers were reelected, and Ander-

son will be the place of meeting in 1937. Efforts will be made to include more largely Georgia and North Carolina, the field from which both teachers and graduates may be interested. This type of refresher course is now in the ascendency in this country and abroad. We have thus made a good start in South Carolina.

SURGERY

WM. H. PRIOLEAU, M.D., F.A.C.S., CHARLESTON, S. C.

"READJUSTMENTS IN THE THORACIC CAGE AND ITS CONTENTS FOLLOWING TOTAL AND PARTIAL PNEUMECTOMY"

Recently additional light has been thrown upon the fate of thoracic cavities resulting from partial or total removal of a lung. Shifting of the mediastinum and the level of the diaphragm, dilatation of the remaining lung tissue, and changes in the conformity and size of the thoracic cavity are the recognized compensatory factors. However it has generally been considered that they are insufficient to effect a complete closure of a cavity of large size. Thus surgical collapse of the chest wall has been commonly resorted to—as a persistent cavity would be an ever present source of danger due to its likelihood of becoming infected. Another factor is that should these compensatory mechanisms succeed in obliterating the cavity, there might be so much displacement of the mediastinum as to interfere with the cardio-respiratory function. For this reason also thoracoplasty has been resorted to—to keep the mediastinum in its proper place.

In a study of 36 cases—10 of total pneumectomy, and 2 of post-traumatic atrophy of the left lung—Dr. William Reinhoff of Baltimore made some important observations concerning the obliteration of large pleural cavities. (Sou. Med. Jour. 29:445 May '36).

The compensatory factors aforementioned are of primary importance and are the first to take effect. The remaining lung tissue becomes greatly dilated—this Dr. Reinhoff distinguishes sharply from emphysema which is a disease process. Cavities on the left, unless very large, are likely to be taken care of completely in this way. The heart is rotated and the right lung extends actually into the left hemithorax both anteriorly and posteriorly. Such is not the condition in cavities on the right. Whereas there is some shifting of the mediastinum, the heart does not rotate as readily to the right, and apparently the left lung can not cross the midline. The result is that the major portion of the cavity has to be cared for in another way.

Immediately following operation there is an outpouring of serosanguinous fluid. The air collects at the top and is rapidly absorbed. The fluid very soon clots. Fibrin is precipitated and a scaffolding forms. Fibroblasts grow in from the periphery and thus organize the contracted clot. The result is a fenestrated labyrinth of connective tissue, with clear fluid occupying the interspaces. On X-ray examination it is difficult to distinguish this from lung parenchyma. It is in this manner that the pleural cavities, not compensated for otherwise, are obliterated.

The foregoing observations, while definite, have not yet been made upon a sufficient number of cases to be accepted as generally obtaining. Should they be, they will have a far-reaching effect upon thoracic surgery, simplifying certain phases of it. The post-operative effusion will be considered as a beneficent mechanism; no longer will aspiration be practiced in these cases to prevent infection.

PATHOLOGICAL CONFERENCE, MEDICAL COLLEGE OF THE STATE OF SOUTH CAROLINA

KENNETH M. LYNCH, M. D., PROFESSOR OF PATHOLOGY

ABSTRACT No. 311 (30742)

Feb. 21, 1936

Case of Dr. Cannon

Student Able (reading):

A negro man, 43 years of age, admitted 1-9-36; died 1-13-36.

History (obtained from friend): Less than an hour before his admission to the hospital, the patient was apparently well and was working. Suddenly he stopped talking (and probably fell), was removed to a bed. 30 minutes later convulsive moments began, and the mouth was drawn to the left side. Later the convulsive movements stopped and the patient was admitted in a comatose condition.

No history of trauma, alcoholism, diabetes, epilepsy or any other illness.

Examination: Temp. 99, pulse 96; resp. 36; B. P. 175-60. A well developed, comatose male. Skin: dry and warm. Eyes: "Rt. eye scarred and opaque (bluish) with tumor of sclera. Left pupil irregular, constricted, does not react." Slight tremor of tongue. Teeth carious. Inguinal lymph glands palpable. Neck: negative. Chest: expansion equal, lungs clear. Heart: apparently not enlarged. Apical impulse diffuse. Systolic murmur (chart infers that murmur was apical). Rate slow, numerous extra-systoles. Abdomen: No abnormalities noted. Neurological: Left extremities slightly spastic, left eye turned up and out. KJ's positive on both sides, no ankle clonus or Babinski. No reaction to painful stimuli on left, reaction present on right.

Lab: Urine (1-11) S. G. 1.030; alb. 0, sugar a trace, acetone 0, casts 0, occasional leukocyte, RBC 0. Blood (1-9) Hb. 90 per cent (T), WBC 11,600, polys 70 per cent, lymphs 26 per cent, monos 4 per cent; plas neg. Thick and thin smear for plas (1-10) neg. Spinal Fluid (1-9): normal pressure, clear, colorless; cells 28 per cu. mm., lymphs predominating; globulin a trace, sugar 1 plus; colloidal gold 1112210000; Kolmer and Kline 4 plus.

Course: Temp. rose suddenly to 106 at 8 A.M. of 1-10, fell to 101.6 at 12 noon, then rose slowly to 106 on 1-12 at 8 A.M., remaining near that level until death. Pulse followed temp. closely. Resp. usually about 40, were 66-68 for last 12 hours. On 1-10 convulsions were generalized and continued at intervals of a few minutes all day. Face drawn to left side. On 1-11, ankle clonus was elicited on left. "Apparently a flaccid paralysis of left side of body." Face continued to twitch, but generalized convulsions ceased, patient remaining in coma. Spinal fluid on 1-11 showed dark, yellow-brown fluid under slightly increased pressure, with a few blood cells present. (Further study of this specimen apparently not made). 1-12 Rales over bases of both lungs. Ceased to breathe on 1-13-36 at 12-45 A.M.

Dr. J. H. Cannon (conducting): Mr. Goodlett, will you open the discussion?

Student Goodlett: We have the record of a negro man, 43 years of age, who had apparently been quite well until just before the onset of his present illness. This onset was very abrupt, and resulted in paralysis and convulsions. Examination disclosed a paralysis of the left side of the body, and of the right side of the face (since the face was drawn to the left). This gives us a crossed paralysis, localizing the lesion in the pons, at the level of the nucleus of the facial nerve. In addition, the cell count in the spinal fluid, and the colloidal gold curve suggest the presence of syphilitic disease of the central nervous system, and the spinal fluid Kolmer and Kline bear this out.

It seems to me, then, that we have a case of syphilitic disease of the meninges and probably of the cerebral blood vessels. I believe that the suddent onset of cerebral symptoms indicated a hemorrhage into the pons, probably on the basis of the vascular disease already present.

Dr. Cannon: You believe that syphilis was the cause of a cerebral hemorrhage. What

else might have caused this clinical picture?

Student Goodlett: He may have had a cerebral embolism from an old heart lesion.

Dr. Cannon: Why do you say an "old" heart lesion? How would embolism occur from an old lesion?

Student Goodlett: Possibly an old arterosclerotic plaque broke off and lodged in the brain. Or, in case of a more recent and an active valvular lesion of the heart, a vegetation might have broken off and lodged there.

Dr. Cannon: Is syphilis a common cause of cerebral hemorrhage?

Student Goodlett: Not a common one, but it causes hemorrhage occasionally.

Dr. Cannon: How about thrombosis? Could it have caused this picture?

Student Goodlett: With thrombosis, the symptoms usually come on more gradually.

Dr. Cannon: Mr. Rutledge, can you add anything to the discussion?

Student Rtuledge: I differ with Mr. Goodlett in one point: I believe that syphilis is a common cause of cerebral hemorrhage. But I agree with him in the general analysis of the case, and my diagnosis is cerebral hemorrhage on a background of meningo-vascular syphilis. I do not believe that the onset could have been as sudden as this in cerebral embolism, and the area involved would hardly have been as large as the record here seems to indicate.

I saw the autopsy, though, and there was certainly no gross evidence of massive hemorrhage; a few small petechiae were noted, but no hemorrhages large enough to give these symptoms.

Dr. Cannon: Do you believe that hemorrhage is a more common manifestation of meningo-vascular syphilis than thrombosis is?

Student Rutledge: No, I believe that thrombosis is more common, but I do not believe that hemorrhage is uncommon.

I think the essential lesion here was located in the pons. That would easily account for the fever, because of its proximity to the heat center. That explanation is hardly necessary, however, as the fever could be due to the convulsions themselves.

Dr. Cannon: Does anyone else care to comment? (No answer).

Dr. Lynch will present the autopsy findings now and leave time for discussion after the autopsy report.

Dr. Lynch: There was little to be seen grossly at autopsy, the diagnosis in a case such as this depending on the histological findings.

You see here a section of brain (using microprojector), showing a medium-sized blood vessel that is greatly dilated and contains a recently formed thrombus. It is surrounded by a well-defined collar of lymphocytes. The same appearance is noted in sections taken from all parts of the brain, indicating that the process was quite diffuse. Numerous petechial hemorrhages can be seen, and these were noted grossly, and given as the finding on which the diagnosis of encephalitis was made grossly. Within the meninges there is a similar picture, as you can see; here the meningeal blood vessels are surrounded by lymphocytic cells. But the meningeal involvement in this case appears minor when compared to the relative severity of the inflammation within the brain. In addition, evidences of degeneration can be made out within the various nerve-cell bodies, although that cannot be demonstrated with a projector.

It is thus easily established as a case of encephalitis. But what is the nature of the encephalitis? It resembles in almost every respect the appearance of the brains from the 1918-19 epidemic of encephalitis. The recent epidemic in St. Louis was usually similar to this, although the lesions were seldom so widespread throughout the brain. Frequently we hear that the type of encephalitis which occurred in the epidemic of 1918-19 is no longer with us. But quite frequently we see a sporadic case which is entirely similar to those other cases. A few years ago we had a number of such cases in close succession here in Charleston, although there were not enough cases to call it an epidemic.

He had positive serological evidence of syphilis, and at autopsy he was found to have a mild syphilitic aortitis. While syphilitic encephalitis is occasionally quite similar to this case on the basis of the microscopic findings, I am inclined to believe that this was a sporadic case of lethargic encephalitis, occurring in a syphilitic individual. The sudden onset in the clinical part of the picture, and the distention of the

blood vessels, the petechial hemorrhages and the marked edema of the brain in the histological part, make me believe that this was not syphilitic.

In addition to his encephalitis, this patient also had an early lobar pneumonia, the lung being in a well-established stage of red hepatization. This would indicate that his pneumonic inflammation probably began at about the time his sudden cerebral symptoms occurred. I am speculating on the possibility that this man had the virus of encephalitis in his brain, but lying latent, and that the acute infection caused the latent cerebral infection to become active. This is the explanation that is commonly given for those cases of typical encephalitis that occur in children following even the milder contagious diseases, as mumps, chicken pox, measles and pertussis. The so-called "post-vaccinal" encephalitis is explained in this manner by some. Of course I do not believe that the pneumococcus itself caused the encephalitis, as the pneumococcus would have caused the suppuration; but I am inclined to believe that pneumonic infection permitted the encephalitis virus to stir itself from a latent stage and become active.

I would like to hear some clinical discussion of the anatomical findings in this case.

Dr. Cannon: Since this patient was on my service, I guess I must try to defend my diagnosis of syphilitic vascular disease of the brain, with hemorrhage. The suddenness of onset is very difficult to correlate with any diagnosis other than hemorrhage. I am afraid that I would make the same error if I had such a case again. But we are taught a worth-while lesson from this case: event if the inflammatory process causing the symptoms is diffuse, there may be localizing symptoms to make one suspect a focal lesion.

Dr. Wilson: I went over this patient on the wards in one of my clinics with the class. We were discussing the different causes of hemiplegia, and decided that this man's hemiplegia was a result of vascular disease and cerebral hemorrhage.

When we err, we should always ask ourselves the question, Can we avoid this error next time? The inconsistency of the findings should have made us suspicious of something unusual in this case, but instead it made us suspect that the original examiner, who made the record, had made incorrect observations. With hemorrhage, spasticity usually comes on several days after the onset of symptoms; here it was present when first seen. Positive ankle clonus is usually present when spasticity has developed; here flaccidity followed spasticity, and the ankle clonus was obtained during the time of flaccidity.

I have seen a number of cases of encephalitis in which a hemiplegia developed, but prior to this case I had never had an autopsy on such a case of encephalitis.

I have seen the symptoms of encephalitis come on very abruptly, but the symptom at onset is usually headache.

The blood pressure in this case would seem to suggest an insufficiency of the aortic valve, because of the high systolic pressure and the low diastolic pressure. Since there was no evidence of disease of the aortic valve at autopsy, I am at a loss to explain this blood pressure reading; possibly it would have been different if it had been taken several times.

Dr. Chamberlain: Since I was not here for the early part of this conference, I could not take a part in the discussion prior to the presentation of the autopsy findings by Dr. Lynch. And my statements now may be somewhat biased.

But there is much in the record to make us question a diagnosis of cerebral hemorrhage, although the suddenness of the onset would certainly make us consider that diagnosis closely. The variation in the reflexes and in the muscular condition of the extremities is certainly not what we would expect in a hemorrhage severe enough to cause coma. Rather we would expect complete flaccidity and a total loss of the reflexes in this stage, as Dr. Wilson pointed out. Convulsions can only be explained by a lesion in or near the motor cortex. But the deviation of the left eye, and that eye only, to the left and upward, suggests that either the left abducens nerve was badly stimulated or else the left oculomotor nerve was paralysed distal to the nucleus. In either case, such a lesion would have to be well below the cortical area. Whether the facial paralysis was of a cortical type or not cannot be determined from the record; if it was not of a cor-

tical type, it would be further evidence of the diffuse character of the lesion.

Since the various parts of the neurological picture cannot be explained on a localized lesion in one part of the brain, it becomes apparent that the lesion must have been a diffuse one, of the nature of an encephalitis or a meningo-encephalitis.

Dr. Lynch has pointed out that occasionally any one of many bacterial agents or their toxins may go haywire and be followed by encephalitis, and the subsequent pictures of encephalitis may be quite alike in all respects, no matter what the inciting agent. Experimentally, an acute type of encephalitis can be caused in the rabbit by inoculating him with the serum from an ordinary fever blister. Dr. Lynch's thesis as to the possible cause of the encephalitis seems to me to be as logical as any.

OBSTETRICS AND GYNECOLOGY

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PRENATAL CLINICS OF THE MATERNAL AND CHILD HEALTH DIVISION OF THE STATE BOARD OF HEALTH

The Division of Maternal and Child Health of the State Board of Health has organized and is conducting prenatal and well children clinics in most of the counties of South Carolina. The director of the division is Dr. R. W. Ball, formerly connected with the county health unit of Richland county. He is deeply interested in the work of his division, and is using every effort to make this work of real benefit not only to the indigent women and children of the State, but to the doctors as well.

The writer has had considerable opportunity to observe the workings of this new division of the State Board. He has conducted a number of the prenatal clinics in the Piedmont section of the State. Dr. R. C. Bruce, president of the State Medical Association, appointed him to represent that body on the Advisory Board of the Division of Maternal and Child Health and he has sat with the board at their first quarterly meeting. Furthermore, he has had a number of conversations and some correspondence with Dr. Ball, the director. These various contacts have enabled him to reach certain conclusions concerning the work as it is now organized, the ultimate aims of the department, the benefits that will accrue to the women of the State, and finally the effect that this work will have on the medical profession.

First of all, it should be realized that the work of the division is in its infancy and that it began without personnel and with a plan which was only in outline. The organization is being improved day by day. The personnel is being trained and improved, and what it is today is no true indication of what it will ultimately be. The work that is now being done will be improved upon, its scope will be broadened to reach more women and children, the educational features will be stressed more and more, and the cost per patient reached will be materially lessened.

The writer's first impression is that the director is earnestly trying to secure the cooperation of the doctors of South Carolina. He is trying to protect their interests, and he is giving every suggestion made by any one of them most careful consideration. He has no desire or intention of taking paying practice away from them, and he wishes to have them to cooperate in the conduct of the clinics. County Medical societies have been asked for suggestions, and these suggestions have been followed so far as practicable since the inauguration of the program.

The clinics are carefully worked up in advance by field workers. The prospective patients are called upon individually, and their financial status is investigated. None but the truly indigent, who would otherwise receive no prenatal care except in the case of catastrophe,

are allowed to attend the clinics. More than this, where defects are found that seem to require medical treatment, no such treatment is given, the clinics being entirely diagnostic. The patient requiring treatment is sent to the physician of her choice with a statement of the defect found, and the case is turned over to him for treatment. Considering the true indigency of these women, the writer feels that this is carried to an extreme, and that the practice tends to add to the burden of the practitioner with little or no chance of remuneration. However, doctors are sensitive of their prerogative. Only recently he heard a doctor who already has to carry a large burden of free practice quoted as having said that if any woman in his practice attended one of these free clinics, she need not call on him to deliver her. Such an attitude is unfortunate. So long as only truly indigent women are admitted to these free clinics, and so long as only those who are receiving and will receive no prenatal care unless they are so admitted are given that privilege, neither the financial return nor the prestige of any doctor will be hurt. These postulates are seemingly being carried out in a meticulously careful manner.

Some criticism is being expressed with regard to the professional personnel of the clinics. One can well understand this, and yet it is the writer's opinion that this would be more quickly and more satisfactorily worked out if the local doctors in those communities where the clinics are being held would show a greater tendency to cooperate in its solution. The attendance of local doctors at the clinics conducted by the writer has in most instances been occasional and brief. There appears to be no general interest in them. One surely could not expect to be assigned the conduct of the clinic until there is evidenced a willingness to cooperate. Already in twelve instances a local doctor has been assigned to well baby clinics, but only seven local doctors, so far as the writer is aware, have shown enough interest to warrant assignment to prenatal clinics. It is hoped that this will soon be remedied. As the plan now is, a doctor becomes eligible for such assignment when he has attended two clinics conducted by a specialist.

The definitely educational side of the program has hardly begun. So far this has been more coincidental than purposeful. The field workers in inviting women to come to the clinics have discussed with them the importance of prenatal care. These conversations set up, as it were, foci from which information, doubtlessly more or less garbled, permeates. The few doctors who have irregularly attended the clinics have seen what constitutes a minimumly adequate prenatal examination. But this is not enough. At every clinic the patients should be given a simple lecture on the hygiene of pregnancy either by the doctor or by the nurse. Furthermore, there should be some effort made at community mass instruction, open not only to the indigent who have been contacted, but to every one. The free clinics should not be mentioned at these meetings, but the stress should be put on the idea of seeing one's doctor early and regularly when pregnant. While the program has been deficient in this respect, plans are already formulated to begin such a program. The writer wonders if a plan could not be worked out so that public meetings could be sponsored by the local county medical societies. The program could be arranged either wholly or in part by Dr. Ball's office. No doubt Dr. Ball would be glad to cooperate in such a plan. If the county society did not care to take up such a program, there is an excellent opportunity here for the woman's auxillary to do something constructive for the community.

This is not truly State medicine. True it is an opening wedge, but one that is still largely under the control of the organized medical profession. Whether it shall continue under such control remains to be seen. It will not and can not be, unless organized medicine shows an active interest in it and assists in its direction and guidance. Quoting from the bulletin, "Working Details of the State Plan for Maternal and Child Clinics," "The responsibility for treatment of abnormalities found at clinics lies with the medical profession. If the local physicians do not treat these patients, then naturally it will ultimately become a State problem. It is not our desire to enter the field of curative medicine; our work is entirely preventive, and with the cooperation of the local physicians it will remain so." Are the doctors of South Carolina too blind to see or too indolent to seize the opportunity that is here?